



# UNITED STATES PATENT AND TRADEMARK OFFICE

*a*  
UNITED STATES DEPARTMENT OF COMMERCE  
United States Patent and Trademark Office  
Address: COMMISSIONER FOR PATENTS  
P.O. Box 1450  
Alexandria, Virginia 22313-1450  
www.uspto.gov

APPLICATION NO.	FILING DATE	FIRST NAMED INVENTOR	ATTORNEY DOCKET NO.	CONFIRMATION NO.
10/607,455	06/26/2003	Paula J. Bates	LOU01-011-US	3317
43320	7590	02/06/2008	EXAMINER	
EVAN LAW GROUP LLC			HUYNH, PHUONG N	
600 WEST JACKSON BLVD., SUITE 625			ART UNIT	PAPER NUMBER
CHICAGO, IL 60661			1644	
MAIL DATE		DELIVERY MODE		
02/06/2008		PAPER		

**Please find below and/or attached an Office communication concerning this application or proceeding.**

The time period for reply, if any, is set in the attached communication.

<b>Office Action Summary</b>	<b>Application No.</b>	<b>Applicant(s)</b>	
	10/607,455	BATES ET AL.	
	<b>Examiner</b>	<b>Art Unit</b>	
	Phuong Huynh	1644	

-- The MAILING DATE of this communication appears on the cover sheet with the correspondence address --  
**Period for Reply**

A SHORTENED STATUTORY PERIOD FOR REPLY IS SET TO EXPIRE three MONTH(S) OR THIRTY (30) DAYS, WHICHEVER IS LONGER, FROM THE MAILING DATE OF THIS COMMUNICATION.

- Extensions of time may be available under the provisions of 37 CFR 1.136(a). In no event, however, may a reply be timely filed after SIX (6) MONTHS from the mailing date of this communication.
- If NO period for reply is specified above, the maximum statutory period will apply and will expire SIX (6) MONTHS from the mailing date of this communication.
- Failure to reply within the set or extended period for reply will, by statute, cause the application to become ABANDONED (35 U.S.C. § 133). Any reply received by the Office later than three months after the mailing date of this communication, even if timely filed, may reduce any earned patent term adjustment. See 37 CFR 1.704(b).

**Status**

- 1) Responsive to communication(s) filed on 20 November 2007.
- 2a) This action is **FINAL**.                            2b) This action is non-final.
- 3) Since this application is in condition for allowance except for formal matters, prosecution as to the merits is closed in accordance with the practice under *Ex parte Quayle*, 1935 C.D. 11, 453 O.G. 213.

**Disposition of Claims**

- 4) Claim(s) 13-16,43,44,46,47 and 51-62 is/are pending in the application.
- 4a) Of the above claim(s) \_\_\_\_\_ is/are withdrawn from consideration.
- 5) Claim(s) \_\_\_\_\_ is/are allowed.
- 6) Claim(s) 13-16,43,44,46,47 and 51-62 is/are rejected.
- 7) Claim(s) \_\_\_\_\_ is/are objected to.
- 8) Claim(s) \_\_\_\_\_ are subject to restriction and/or election requirement.

**Application Papers**

- 9) The specification is objected to by the Examiner.
- 10) The drawing(s) filed on \_\_\_\_\_ is/are: a) accepted or b) objected to by the Examiner.  
 Applicant may not request that any objection to the drawing(s) be held in abeyance. See 37 CFR 1.85(a).  
 Replacement drawing sheet(s) including the correction is required if the drawing(s) is objected to. See 37 CFR 1.121(d).
- 11) The oath or declaration is objected to by the Examiner. Note the attached Office Action or form PTO-152.

**Priority under 35 U.S.C. § 119**

- 12) Acknowledgment is made of a claim for foreign priority under 35 U.S.C. § 119(a)-(d) or (f).
- a) All    b) Some \* c) None of:
  1. Certified copies of the priority documents have been received.
  2. Certified copies of the priority documents have been received in Application No. \_\_\_\_\_.
  3. Copies of the certified copies of the priority documents have been received in this National Stage application from the International Bureau (PCT Rule 17.2(a)).

\* See the attached detailed Office action for a list of the certified copies not received.

**Attachment(s)**

- 1) Notice of References Cited (PTO-892)
- 2) Notice of Draftsperson's Patent Drawing Review (PTO-948)
- 3) Information Disclosure Statement(s) (PTO/SB/08)  
 Paper No(s)/Mail Date 9/11/07; 8/29/07.
- 4) Interview Summary (PTO-413)  
 Paper No(s)/Mail Date. \_\_\_\_\_.
- 5) Notice of Informal Patent Application
- 6) Other: \_\_\_\_\_.

### **DETAILED ACTION**

1. Claims 13-16, 43-44, 46-47, and 51-62 are pending and are being acted in this Office Action.
2. Claim 14 is objected to because “a tumor, a cancer” is redundant.
3. Claim 15 is objected to because said claim should depend from claim 14.
4. Claim 53 is objected to because said claim should depend from claim 52.
5. The following is a quotation of the first paragraph of 35 U.S.C. 112:

The specification shall contain a written description of the invention, and of the manner and process of making and using it, in such full, clear, concise, and exact terms as to enable any person skilled in the art to which it pertains, or with which it is most nearly connected, to make and use the same and shall set forth the best mode contemplated by the inventor of carrying out his invention.

6. Claims 13-16, 43-44, 46-47, and 51-62 are rejected under 35 U.S.C. 112, first paragraph, because the specification, while being enabling only for (1) a method of detecting excessive apoptosis in a subject, comprising preparing a blood sample from the subject, removing cells from said sample, disrupting the apoptotic bodies in said sample, reacting the sample with an antibody that binds specifically to nucleolin and detecting the binding of the antibody to nucleolin in the apoptotic bodies of said sample is indicative of excessive apoptosis in the subject, and (2) a method of detecting excessive apoptosis in a subject, comprising preparing a blood sample from the subject, removing cells from said sample, disrupting the apoptotic bodies in said sample, reacting the sample with an antibody that binds specifically to PARP-1 and detecting the binding of the antibody to PARP-1 in the apoptotic bodies of said sample is indicative of excessive apoptosis in the subject, **does not** reasonably provide enablement for methods of detecting excessive apoptosis as set forth in claims 13-16, 43, 46-47, 51-55, and 57-62 without disrupting the apoptotic bodies. The specification does not enable any person skilled in the art to which it pertains, or with which it is most nearly connected, to make and use the invention commensurate in **scope** with these claims.

Factors to be considered in determining whether undue experimentation is required to practice the claimed invention are summarized *In re Wands* (858 F2d 731, 737, 8

USPQ2d 1400, 1404 (Fed. Cir. 1988)). The factors most relevant to this rejection are the scope of the claim, the amount of direction or guidance provided, the lack of sufficient working examples, the unpredictability in the art and the amount of experimentation required to enable one of skill in the art to practice the claimed invention. The specification disclosure is insufficient to enable one skilled in the art to practice the invention as broadly claimed without an undue amount of experimentation.

The claims encompass methods of detecting excessive apoptosis in a subject, comprising preparing a blood sample from the subject, removing cells from said sample, disrupting the apoptotic bodies in said sample, reacting the sample with any antibody that binds specifically to nucleolin or PARP-1 and detecting the binding of the antibody to nucleolin or PAPR-1 in the apoptotic bodies of said sample is indicative of excessive apoptosis in the subject.

Enablement is not commensurate in scope with detecting the binding of any antibody to any nucleolin or any PAPR-1 *in* any apoptotic bodies of a blood sample from any subject such as any mammals.

In order to detect nucleolin or PAPR-1 *in* the apoptotic bodies (emphasis added), the specification discloses disrupting the apoptotic bodies *before* detecting nucleolin and/or PARP-1 with antibodies to such, see page 20.

The specification discloses both nucleolin and PARP-1 are nuclear proteins located in the S-100 fraction, see page 35. The specification discloses detection of nucleolin shed from U937 cells (human promyeloid cell line) during apoptosis after irradiated with UV in the absence or presence of 3-ABA, see page 39. The specification at page 38 discloses there was a redistribution of nucleolin in the apoptotic nuclei (UV treated or CPT treatment) and nuclear nucleolin staining decreased in the plasma membrane and nuclei. There is no nexus between detecting nucleolin or PARP-1 in apoptotic bodies of U937 cells induced apoptosis with UV or PARP-1 inhibitor 3-aminobezamide (3-ABA) and detecting nucleolin or PARP-1 in apoptotic bodies from serum or plasma of any subject, any mammal such as monkey, dog, cat, rabbit, cow, pig, goat, sheep, horse, rat, mouse, guinea pig, etc, whale, or any human having any diseases such as ones recited in claims 14-16, and 52-54. The specification defines the term subject is a vertebrate, a mammal includes monkey, dog, cat, rabbit, cow, pig, goat, sheep, horse, rat, mouse, guinea pig, whale, human etc, see page 10, last paragraph. The specification discloses only anti-nucleolin antibodies that bind to frog, human and dog, see page 12 and anti-PARP antibodies that bind to mouse and human PARP-1, see page 13.

The state of the art as exemplified by Deng et al (Molecular Biology Reports 23: 191-195, 1996; PTO 1449) who teach nucleolin is one of the most abundant nucleolar protein found in the membrane of nucleoli (see page 191, col. 1, page 193, col. 2, in particular). Deng et al teach the expression of plasma membrane is not universal, even among tissue culture cells, for example, nucleolin is present on the surface of the HepG2 cells (human hepatocarcinoma) but not on lymphoblastoid cells including Molt-4 (human T cell lymphoma) and Wil 2 (see page 194, col. 2, in particular). Deng et al further teach monoclonal antibody such as D3 that binds specifically to human nucleolin but not to mouse (see page 191, col. 2, Materials and methods, in particular).

Given the numerous antibodies to nucleolin and PARP-1, there is insufficient guidance and working example as to the binding specificity of such antibodies.

Given the numerous diseases in any and all vertebrate subjects, and the heterogeneous expression of nucleolin even in cancer cells, there is no nexus between detecting nucleolin in apoptotic bodies of human U937 cells induced apoptosis with UV or PARP-1 inhibitor 3-aminobezamide (3-ABA) and detecting nucleolin in apoptotic bodies from serum or plasma of any subject, any mammal such as monkey, dog, cat, rabbit, cow, pig, goat, sheep, horse, rat, mouse, guinea pig, human, whale, etc having any diseases such as any and all neurodegenerative diseases, any autoimmune diseases other than SLE, any cancer other than endocervical adenocarcinoma, prostatic carcinoma, breast cancer and non-small cell lung carcinoma. It is unpredictable detection of nucleolin or PARP-1 in the apoptotic bodies of plasma or serum or blood sample of any subject is a useful marker for such diseases.

For these reasons, it would require undue experimentation of one skilled in the art to practice the claimed invention. See page 1338, footnote 7 of Ex parte Aggarwal, 23 USPQ2d 1334 (PTO Bd. Pat App. & Inter. 1992).

In re wands, 858 F.2d at 737, 8 USPQ2d at 1404 (Fed. Cir. 1988), the decision of the court indicates that the more unpredictable the area is, the more specific enablement is necessary. In view of the quantity of experimentation necessary, the limited working examples, the unpredictability of the art, the lack of sufficient guidance in the specification and the breadth of the claims, it would take an undue amount of experimentation for one skilled in the art to practice the claimed invention.

7. The following is a quotation of the appropriate paragraphs of 35 U.S.C. 103(a) that form the basis for the rejections under this section made in this Office Action:

A person shall be entitled to a patent unless:

(a) A patent may not be obtained though the invention is not identically disclosed or described as set forth in section 102 of this title, if the differences between the subject matter sought to be patented and the prior art are such that the subject matter as a whole would have been obvious at the time the invention was made to a person having ordinary skill in the art to which said subject matter pertains. Patentability shall not be negated by the manner in which the invention was made.

8. This application currently names joint inventors. In considering Patentability of the claims under 35 U.S.C. 103(a), the examiner presumes that the subject matter of the various claims was commonly owned at the time any inventions covered therein were made absent any evidence to the contrary. Applicant is advised of the obligation under 37 CFR 1.56 to point out the inventor and invention dates of each claim that was not commonly owned at the time a later invention was made in order for the examiner to consider the applicability of 35 U.S.C. 103(c) and potential 35 U.S.C. 102(e), (f) or (g) prior art under 35 U.S.C. 103(a).
  
9. Claims 13-16, 43-44, 46, 51-57 and 59-62 are rejected under 35 U.S.C. 103(a) as being unpatentable over Holdenrieder et al (Int J Cancer 95: 114-120, March 2001; PTO 892) in view of Martelli et al (of record, J cellular Biochemistry 78: 264-277, 2000; PTO 892).

Holdenrieder et al teach a method of detecting excessive apoptosis in serum of patient with benign and malignant disease by detecting nucleosomes that are packed into apoptotic bodies (see entire document, col. 1, in particular). The reference method comprises the step of preparing a blood sample from a subject such as a human (mammal) having various diseases such as breast cancer, lung cancer, lymphoma, autoimmune inflammatory disease such as colitis, inflammatory disease such as pancreatitis and removing cells from the sample by centrifugation to collect serum or plasma (see page 115, col. 1, Patients, in particular), and reacting the serum or plasma with antibody that binds to nucleosomes and detecting the binding of anti-nucleosome antibody to nucleosome in ELISA assays (see page 114-115, Material and Methods, in particular). Holdenrieder et al teach the samples containing apoptotic bodies were homogenized (disrupting the apoptotic bodies) and diluted 1: 4 with incubation buffer (see page 115, second full paragraph, in particular). Holdenrieder et al teach the advantage of using serum or plasma for quantifying excessive apoptosis is that it is non invasive and easily perform method; it could be applied in daily routine and the concentration of nucleosomes in the serum reflects a snapshot of the rate of cell death at a defined time in patient before and after therapy (see page 114, col. 2, page 119, in particular).

The invention in claim 13 differs from the teachings of the reference only in that the method of detecting excessive apoptosis by reacting the sample with an antibody that binds specifically to nucleonin in apoptotic bodies instead of nucleosome in apoptotic bodies.

The invention in claim 51 differs from the teachings of the reference only in that the method of detecting excessive apoptosis by reacting the sample with an antibody that binds specifically to PARP-1 to in apoptotic bodies instead of nucleosome in apoptotic bodies.

Martelli et al teach a method of detecting apoptosis comprising preparing a sample from which cells such as HL60 have been removed from tissue culture (see page 265, col. 1, Materials and methods, Cell Culture and Induction of Apoptosis, in particular), detecting nucleolin using monoclonal antibody that binds to protein C23/nucleolin and monoclonal antibody such as C-2-10 that binds to PARP from Oncogene Research Products (see page 265, paragraph bridging col. 1 and col. 2, page 269, col. 2, fourth paragraph, Figure 7, in particular). Martelli et al further teach the method further comprises membrane disruption by lysing the cells in lysis buffer (see page 266, col. 1, Polyacrylamide Gel Electrophoresis and Immunoblotting of Cell Lysates, page 275, Figure 9, in particular). Martelli et al further teach apoptosis can be detected using antibody that binds to protein C23/nucleolin and/or antibody such as C-2-10 that binds to PARP and the increase rate of apoptosis are responsible for various disease such as degenerative disease, autoimmune disease, and carcinoma (see page 264, col. 1, in particular).

Therefore, it would have been obvious to one of ordinary skill in the art at the time the invention was made to substitute the antibody that binds to nucleosome in apoptotic bodies in the method of detecting excessive apoptosis in a subject of Holdenrieder et al for the monoclonal antibody that binds to nucleonin or monoclonal antibody that binds to PARP-1 for a method of detecting apoptosis as taught by Martelli et al.

One having ordinary skill in the art would have been motivated to do this because Holdenrieder et al teach the advantage of using serum or plasma for quantifying excessive apoptosis in a subject is that the method is non-invasive and easily perform; it could be applied in daily routine and the concentration of nucleosomes in apoptotic bodies of the serum reflects a snapshot of the rate of cell death at a defined time in patient before and after therapy (see page 114, co.. 2, in particular). Martelli et al teach apoptosis can be detected using any antibody that binds to protein C23/nucleolin and/or antibody such as C-2-10 that binds to PARP; the increase rate of apoptosis are responsible for various disease such as degenerative disease, autoimmune disease, and carcinoma (see page 264, col. 1, in particular). From the combined teachings of the

references, it is apparent that one of ordinary skill in the art would have had a reasonable expectation of success in producing the claimed invention.

10. Claim 47 is rejected under 35 U.S.C. 103(a) as being unpatentable over Holdenrieder et al (Int J Cancer 95: 114-120, March 2001; PTO 892) in view of Martelli et al (of record, J cellular Biochemistry 78: 264-277, 2000; PTO 892) as applied to claims 13-16, 43-44, 46, 51-57 and 59-62 mentioned above and further in view of Hanakahi et al (Proc Natl Acad Sci 94: 3605-3610, 1997; PTO 892).

The combined teachings of Holdnrieder et al and Matelli et al have been discussed *supra*.

The invention in claim 47 differs from the teachings of the references only in that the method of detecting excessive apoptosis in a subject wherein the antibody to nucleolin is a polyclonal antibody instead of a monoclonal antibody.

Hanakahi et al teach a recombinant human nucleolin as well as polyclonal antibody that binds to human nucleolin (see page 3607, Fig 2, in particular). The reference polyclonal antibody is specific to the human nucleolin since it detects a single band by Western blot (see 3607, Figure 2B, in particular).

Therefore, it would have been obvious to one of ordinary skill in the art at the time the invention was made to substitute the monoclonal antibody that binds to nucleolin of Martelli et al in the method of detecting excessive apoptosis in a subject of Holdenrieder et al for the polyclonal antibody that binds specifically to nucleolin as taught by Hanakahi et al.

One having ordinary skill in the art would have been motivated to substitute monoclonal for the polyclonal antibody that binds to nucleolin this because the polyclonal antibody to nucleolin of Hanakahi et al is quite specific as it detects a single band by Western blot (see 3607, Figure 2B, in particular). From the combined teachings of the references, it is apparent that one of ordinary skill in the art would have had a reasonable expectation of success in producing the claimed invention.

11. Claim 58 is rejected under 35 U.S.C. 103(a) as being unpatentable over Holdenrieder et al (Int J Cancer 95: 114-120, March 2001; PTO 892) in view of Martelli et al (of record, J cellular Biochemistry 78: 264-277, 2000; PTO 892) as applied to claims 13-16, 43-44, 46, 51-57 and 59-62 mentioned above and further in view of Solani et al (Eur J Histochem 45: 389-392, 2001; PTO 892).

The combined teachings of Holdnrieder et al and Matelli et al have been discussed supra.

The invention in claim 58 differs from the teachings of the references only in that the method of detecting excessive apoptosis in a subject wherein the antibody to PARP-1 is a polyclonal antibody instead of a monoclonal antibody.

Soldani et al teach a method of detecting apoptosis using polyclonal antibody that binds to PARP-1 (see page 390, col. 1 Materials and Methods, in particular). Soldani et al teach PARP-1 is detected in the same cell using TUNEL assay, which is another assay for detecting apoptosis (see page 391, Fig 1 d, h, in particular). Soldani et al teach in autoimmune disease such as SLE, there is a defective clearance of apoptotic bodies and it is the accumulation of apoptotic bodies that could trigger the production of autoantibodies against nuclear components expressed on the apoptotic bodies (see page 390, col. 2, last paragraph, in particular).

Therefore, it would have been obvious to one of ordinary skill in the art at the time the invention was made to substitute the monoclonal antibody that binds to PARP-1 of Martelli et al in the method of detecting excessive apoptosis in a subject of Holdenrieder et al for the polyclonal antibody that binds to PARP-1 as taught by Soldani et al.

One having ordinary skill in the art would have been motivated to substitute the monoclonal antibody for the polyclonal antibody that binds to PARP-1 because the polyclonal antibody of Solani et al has been shown to detect apoptosis and confirm using the DNA fragmentation TUNEL assay which can be visualized using double immunofluorescence detection assays (see page 391, Fig 1 d, h, in particular). From the combined teachings of the references, it is apparent that one of ordinary skill in the art would have had a reasonable expectation of success in producing the claimed invention.

12. Claims 13, 14, 16, 51, 52-52 and 54 rejected under 35 U.S.C. 103(a) as being unpatentable over Holdenrieder et al (Int J Cancer 95: 114-120, March 2001; PTO 892) in view of Martelli et al (of record, J Cellular Biochemistry 78: 264-277, 2000; PTO 892) as applied to claims 13, 15-16, 43-44, 46, 51, 53-57 and 59-62 mentioned above and further in view of Gougeon et al (J Immunology 156: 3509-3520, 1996; PTO 892) or Andrade et al (Apoptosis in Systemic Lupus Erythematosus", Rheumatic Disease Clinics of North America vol, 2, pages 215-227, May 2000; PTO 1449) or Aihara et al (Human Pathology 25(8): 797-801, 1994; PTO 1449).

The combined teachings of Holdnrieder et al and Matelli et al have been discussed supra.

The invention in claim 14 and 52 differs from the teachings of the references only in that the method of detecting excessive apoptosis in a subject wherein the subject having Acquired Immunodeficiency Syndrome or an autoimmune disease or prostatic carcinoma.

Gougeon et al teach patients infected with HIV having Acquired Immunodeficiency Syndrome have increased apoptosis of peripheral lymphocytes (see entire document, abstract, page 3517, in particular). Significant correlation was found between the intensity of spontaneous and activation-induced apoptosis in total lymphocytes and disease progression (see page 3517, col. 2, in particular).

Andrade et al teach patients with autoimmune disease such as systemic lupus erythematosus (SLE) have defects in clearance and degradation of apoptotic corpse (apoptotic bodies) in these patients (see page 221-222, in particular). Because of the defect in the clearance of apoptotic bodies and autoantigens clustered and concentrated on the surface blebs of apoptotic cells led to the generation of autoantibodies that recognize the surface of the apoptotic cells (see page 217 and 219, last paragraph, in particular).

Aihara et al teach the frequency of apoptotic bodies correlates with higher Gleason Grade in prostate cancer, which is predictive of tumor progression (see abstract, Figure 2, in particular).

Therefore, it would have been obvious to one of ordinary skill in the art at the time the invention was made to substitute the cancer patient in the method of detecting excessive apoptosis of Holdnrieder et al and Matelli et al for the patient with Immunodeficiency Syndrome as taught by Gougeon et al, or the patient with autoimmune disease such as systemic lupus erythematosus (SLE) as taught by Andrade et al or the patient with prostate cancer as taught by Aihara et al.

One having ordinary skill in the art would have been motivated to substitute the patient because Gougeon et al teach patients infected with HIV having Acquired Immunodeficiency Syndrome have increased apoptosis of peripheral lymphocytes (see entire document, abstract, page 3517, in particular). Andrade et al teach that patients with autoimmune systemic lupus erythematosus (SLE) have increased apoptosis and the defects in clearance and degradation of apoptotic corpse (apoptotic bodies) that led to autoimmunity in these patients (see page 221-222, in particular). Aihara et al teach the frequency of apoptotic bodies correlates with higher Gleason Grade in prostate cancer, which is predictive of tumor progression (see abstract, Figure 2, in particular). One having ordinary skill in the art would have been motivated to detect excessive apoptosis in patient because Holdenrieder et al teach the advantage of using serum or plasma for quantifying excessive apoptosis in a subject is that the method is non-invasive and easily perform;

it could be applied in daily routine and the concentration of apoptotic bodies in the serum reflects a snapshot of the rate of cell death at a defined time in patient before and after therapy (see page 114, col. 2, in particular). Martelli et al teach apoptosis can be detected using any antibody that binds to protein C23/nucleolin and/or antibody such as C-2-10 that binds to PARP; the increase rate of apoptosis are responsible for various disease such as degenerative disease, autoimmune disease, and carcinoma (see page 264, col. 1, in particular). From the combined teachings of the references, it is apparent that one of ordinary skill in the art would have had a reasonable expectation of success in producing the claimed invention.

13. No claim is allowed.
14. Any inquiry concerning this communication or earlier communications from the examiner should be directed to Phuong Huynh, Ph.D. whose telephone number is (571) 272-0846. The examiner can normally be reached Monday through Thursday from 9:00 a.m. to 6:30 p.m. and alternate Friday from 9:00 a.m. to 5:30 p.m. A message may be left on the examiner's voice mail service. If attempts to reach the examiner by telephone are unsuccessful, the examiner's supervisor, Eileen B O'Hara can be reached on (571) 272-0878. The IFW official Fax number is (571) 273-8300.
15. Any information regarding the status of an application may be obtained from the Patent Application Information Retrieval (PAIR) system. Status information for published applications may be obtained from either Private PAIR or Public PAIR. Status information for unpublished applications is available through Private PAIR only. For more information about the PAIR system, see <http://pair-direct.uspto.gov>. Should you have questions on access to the Private PAIR system, contact the Electronic Business Center (EBC) at 866-217-9197 (toll-free).

/Phuong Huynh/  
Patent Examiner  
Technology Center 1600  
February 1, 2008